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INFLUENCE OF CENTRAL VENOUS PRESSURE UPON
SINUS NODE RESPONSES TO ARTERIAL BAROREFLEX
STIMULATION IN MAN

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Running head: Modulation of baroreflex control of heart rate

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ABSTRACT

There is considerable evidence that the level of afferent cardiopulmonary receptor activity modulates sinus node responses to arterial baroreflex stimulation in experimental animals. We tested the hypothesis that this reflex interaction occurs also in man by measuring sinus node responses to arterial baroreceptor stimulation with phenylephrine injection or neck suction, before and during changes of central venous pressure provoked by lower body negative pressure or leg and lower trunk elevation. Variations of central venous pressure between 1.1 and 9.0 mmHg did not influence arterial baroreflex mediated bradycardia. Baroreflex sinus node responses were augmented by intravenous propranolol, but the level of responses after propranolol was comparable during the control state, lower body negative pressure, and leg and trunk elevation. Sinus node responses to very brief baroreceptor stimuli applied during the transitions of central venous pressure also were comparable in the three states. We conclude that physiological variations of central venous pressure do not influence sinus node responses to arterial baroreceptor stimulation in man.

INTRODUCTION

There is mounting evidence that cardiopulmonary receptors modulate reflex control of the circulation (21,23,24,27,29,36).

Several studies conducted in experimental animals suggest that cardiopulmonary receptor activity modulates arterial baroreflex responses. Koike and co-workers (23) showed that transection of cardiopulmonary vagal afferent nerves in anesthetized dogs augments vasoconstriction provoked by carotid sinus hypotension. Vatner and associates (35) showed that rapid intravenous infusions of saline in conscious dogs increase right atrial pressure and reduce the reflex bradycardia caused by a rise of arterial pressure.

Eckberg, Abboud and Mark (12) showed that after beta-adrenergic blockade, upright posture (which also lowers central venous pressure) augments arterial baroreflex mediated bradycardia. It was speculated that this augmentation might have resulted from decreases in central venous pressure and in tonic inhibition from cardiopulmonary receptors.

In the present experiments, we tested the hypothesis that variations of central venous pressure within a physiological range of man modulate sinus node inhibition caused by brief arterial baroreceptor stimulation. Central venous pressure was altered by lower body negative pressure at 20 mmHg or leg and trunk elevation, and arterial baroreceptors were stimulated by bolus intravenous injections of phenylephrine or by neck suction.

METHODS

Subjects

Volunteers comprised seven healthy men whose average age was 23 ± 4.2 (mean \pm SEM) years. Subjects were studied in the supine position in a post-absorptive state. The University of Iowa Committee on Research Involving Humans approved the project, and all subjects gave their written consent to participate.

Measurements

Polyethylene catheters were inserted into a brachial artery and an antecubital vein after superficial injection of a local anesthetic. The venous catheter was advanced into an intrathoracic vein. Arterial and central venous pressures were measured with Statham pressures transducers. R-R intervals were measured from the electrocardiogram. Forearm blood flow was measured with a mercury-in-silastic strain gauge plethysmograph (17). All measurements were transcribed by an eight-channel, ink-writing recorder.

Arterial Baroreceptor Stimulation

Two methods were used to assess arterial baroreflex control of sinus node function.

In the first method, arterial pressure was raised acutely by bolus intravenous injections of phenylephrine. Each R-R interval, beginning with the rise of arterial pressure, was plotted as a function of the preceding systolic pressure. This relation was analyzed by least squares linear regression, and the reflex control of R-R interval was expressed as the slope of the regression line (31). This slope was accepted for subsequent analysis only if the correlation coefficient were more than 0.80. Several measurements were made during each intervention and the average value was used in this study. Measurements were made during held expiration to reduce the influence of respiration upon arterial baroreflex responses (14). During phenylephrine-induced transient hypertension, arterial pressure and the electrocardiogram were recorded at a paper speed of 50 mm per second.

In the second method, carotid baroreceptors were stimulated with suction applied to a neck chamber (13). With this chamber, pressure around the anterior neck can be reduced rapidly to increase carotid transmural pressure (9), stretch the carotid sinuses (22), and provoke reflex cardiac slowing (12,15). The use of this technique allowed us to measure the carotid baroreflex control of heart rate acutely, as central venous pressure was rising or falling. Suction of 30 mmHg was applied for 0.6 sec, and was begun 0.8 sec before the next anticipated P wave (10). This intensity of neck suction was used because it lies on the linear portion of the stimulus-response relation (10). R-R interval prolongation, from control, was measured from the interval

in which neck suction was begun by a digital computer, in real time. The maximal prolongation of the R-R interval occurred in the first cycle following neck suction. An earlier study showed that neck suction does not lower arterial pressure within this short period (11). Measurements were made at least five times during each intervention and the average value was used in this study.

All subjects remained in sinus rhythm during injections of phenylephrine and during neck suction. Since P-R intervals did not change, R-R intervals were used to define sinoatrial function.

Alteration of Cardiopulmonary Receptor Activity

Cardiopulmonary baroreceptor activity was altered with 1) lower body negative suction at 20 mmHg, and 2) elevation of the legs and lower trunk. We gauged the intensity of the stimulus to cardiopulmonary receptors with measurements of central venous pressure. Lower body negative pressure of 20 mmHg decreases central venous pressure without altering arterial systolic or mean pressure (36).

Protocols

Arterial baroreflex control of sinus node function was measured with the phenylephrine technique in the control state, during lower body negative pressure, and during elevation of the legs and trunk. The bolus injection of phenylephrine was given after central venous pressure stabilized. In three subjects, this protocol was repeated after intravenous

propranolol, 0.2 mg/kg. This was given to minimize the possibility that increased efferent sympathetic activity during lower body negative pressure might obscure a central interaction between cardiopulmonary and arterial baroreflexes (12).

In five subjects, R-R interval prolongation caused by neck suction was measured during the early, dynamic phase, as well as during the stable phase of central venous pressure change produced by lower body negative pressure or leg and trunk elevation. This protocol was used because an interaction between cardiopulmonary and arterial baroreflexes might not be apparent during chronic changes of central venous pressure because of rapid adaptation of cardiopulmonary receptors (7).

Data Analysis

We used the analysis of variance and Dunnett's test for statistical analysis (32). Values of $p \leq 0.05$ were considered significant. Results are expressed as the mean \pm 1 SEM.

RESULTS

Control measurements were obtained before lower body negative pressure and were repeated in five subjects before elevation of legs and trunk (Table 1). Systolic blood pressure was slightly, but not significantly, higher in the second control period as compared with that in the first control. The finding that systolic pressure was slightly higher in the second control period was presumably related to repeated injection of phenylephrine.

Central venous pressure, forearm blood flow and heart rate were not different in the two control periods. The slope of arterial baroreflex control of R-R interval was also comparable in the two control periods.

Lower body negative pressure of 20 mmHg reduced ($p < 0.01$) central venous pressure from 6.0 ± 0.9 to 1.1 ± 1.0 mmHg (Table 1). Forearm blood flow fell ($p < 0.01$) from 4.6 ± 0.4 to 2.9 ± 0.2 ml/min/100gm, and heart rate and systolic arterial pressure did not change significantly from control (Table 1). The slope of arterial baroreflex control of R-R interval after phenylephrine injection was comparable before and after reduction of central venous pressure (Table 1).

Leg and trunk elevation increased ($p < 0.05$) central venous pressure (Table 1). Forearm blood flow, heart rate and systolic pressure did not change significantly from results in preceding control period (Table 1). The slope of arterial baroreflex responses was not altered by elevation of central venous pressure (Table 1).

Propranolol decreased base line heart rate by an average of 11 beats/min and increased arterial baroreflex bradycardia at each level of central venous pressure (Figure 1). However, arterial baroreflex bradycardia after propranolol was comparable during the control state, lower body negative pressure and leg and trunk elevation (Figure 1). Propranolol did not alter systolic arterial pressure or central venous pressure.

The R-R interval prolongation caused by neck suction was comparable during the control state, during the early, dynamic phase, and during steady-state changes of central venous pressure provoked by lower body negative pressure or leg and trunk elevation (Figure 2).

DISCUSSION

Our findings suggest that central venous pressure variations within a physiological range do not alter sinus node responses to arterial baroreceptor stimulation in conscious man. We asked three questions regarding the methods and findings in this study: First, were the provoked changes of central venous pressure sufficiently large to alter cardiopulmonary receptor activity? Second, were the methods used to assess arterial baroreflex control sufficiently sensitive to detect a subtle reflex interaction? Third, was an interaction present, but obscured by other factors?

Was cardiopulmonary receptor activity altered? In most earlier studies of arterial and cardiopulmonary baroreflex interactions, afferent cardiopulmonary activity was eliminated completely by transection (23,24) or cold block (24,27) of the vagus nerves or augmented by massive intravenous infusions (35). The interventions we used in this human study were more subtle; however, the provoked changes of central venous pressure may

have been closer to those which occur physiologically, and probably were sufficient to alter cardiopulmonary receptor activity. Similar changes in cardiac filling pressures have been reported to alter cardiopulmonary vagal afferent activity in experimental animals (34). Moreover, findings in this and earlier studies (21,29,36) suggest that cardiopulmonary baroreceptor activity is altered by lower body negative pressure and elevation of legs and trunk in man. Decreases in central venous pressure during lower body negative pressure at 20 mmHg produced significant forearm vasoconstriction. This vasoconstriction occurs in the absence of changes in arterial systolic and mean pressure and heart rate and presumably originates in cardiopulmonary receptors (36). It is not possible from this and earlier studies (21,29,36) to exclude completely a contribution of reflexes originating in other visceral or somatic receptors. However, previous studies (21,29,36) have been interpreted as indirect evidence that changes in central venous pressure within the physiological range alter cardiopulmonary baroreceptor activity.

Were measurements of arterial baroreflex responses sensitive?

We used two methods to stimulate arterial baroreceptors: bolus intravenous injections of phenylephrine and neck suction. The cardiac slowing provoked by both methods is highly reproducible (10,18) and its magnitude may be altered by other physiological interventions, including exercise (6), sleep (31), respiration (14),

and standing (12). It is unlikely that these techniques are too insensitive to detect an arterial-cardiopulmonary baroreflex interaction since they were successfully used to detect other physiological reflex interactions in earlier studies.

Was an interaction masked by other factors? Several factors may have obscured an influence of the level of cardiopulmonary receptor activity upon arterial baroreflex responses. First, modulation of arterial baroreflex responses may have resulted from lower body negative pressure or leg and trunk elevation, but was very transient because of rapid adaptation of cardiopulmonary receptors (7). The responses to brief neck suction during the rise or fall of central venous pressure preclude this possibility. These transition periods are probably analogous to the period of ramp stimulation used by Chapman and Pankhurst (7) which was accompanied by steadily changing levels of cardiopulmonary receptor activity.

Second, sinus node responses to arterial baroreceptor stimulation might have been augmented by lower body negative pressure, but this interaction was obscured by simultaneous increases of the level of beta-adrenergic opposition to cholinergic bradycardia (12). This possibility seems unlikely because in three subjects given propranolol, arterial baroreflex responses were not altered by lower body negative pressure or leg and trunk elevation (Figure 1).

Third, a central interaction may have been missed because the arterial baroreflex stimuli used also altered afferent cardiopulmonary baroreceptor activity; phenylephrine injections increase left ventricular systolic pressure, and neck suction lowers arterial pressure and left ventricular systolic pressure. Our use of very brief neck suction to stimulate arterial baroreceptor removes this theoretical concern. Sinus node responses to this stimulus occurred during the same cardiac cycle in which it was applied, before the reflex change of aortic or left ventricular pressure could have occurred (11).

Pickering and co-workers (28) and Eckberg (10) showed that the magnitude of human baroreceptor responses varies inversely with heart rate. Accordingly, we were concerned that changes of baseline heart rate caused by lower body negative pressure or leg and trunk elevation might independently alter arterial baroreflex responses, and obscure a true central reflex interaction. This problem did not materialize, however, because changes of baseline heart rate during changes of central venous pressure were negligible.

Bevegård and his co-workers (4) have used sinusoidal neck suction and lower body negative pressure to explore an interaction between arterial and cardiopulmonary baroreceptor reflexes in man. These authors found that the heart rate response to neck suction was not altered, but that the peak-to-peak fluctuations of arterial pressure in response to sinusoidal neck suction between 10 and 40 mmHg were increased by 40 mmHg lower body

negative pressure. In their study, lower body negative pressure at 40 mmHg increased vascular resistance (3). Based on previous studies (26) one would expect that the vasodilator and vasodepressor response to a given level of sympathetic withdrawal provoked by neck suction would be greater at higher baseline vascular resistance. Thus, the apparent augmentation of carotid baroreflex mediated decreases in vascular resistance and arterial pressure might not have involved a true central reflex interaction. The sinus node responses in their study are also difficult to interpret since the fall in arterial pressure with neck suction was greater during lower body negative pressure than it was in control state. Thus, the aortic hypotension which inhibits aortic baroreceptors and opposes stimulation of carotid baroreceptor stimulation was greater than in the control state. Because of these considerations, it is difficult to interpret their observations in terms of a central interaction of cardiopulmonary and carotid reflexes.

We might compare briefly heart rate response to the increase in central venous pressure in experimental animals and humans. Bainbridge described an increase in heart rate during intravenous infusion of saline in dogs and attributed tachycardia to reflex withdrawal of vagal tone (2). Recent studies have confirmed that reflex tachycardia occurs even with a small increase in arterial pressure during intravenous infusion of saline in conscious dogs (20,35). Several mechanisms have been proposed to explain the

reflex tachycardia during infusion. Vatner, et al (35), have suggested that vagal afferent input from cardiopulmonary baroreceptors may be involved in the modulation of the arterial baroreflex control of heart rate. Stinnett, et al (33), suggested that vagal afferent pathways are not involved in modulation of arterial baroreceptor control of heart rate during intravenous infusion of saline. Other studies (5,19) have suggested that spinal mechanisms may contribute to reflex tachycardia during intravenous infusion.

The results of our study suggest that reflex tachycardia and modulation of arterial baroreflex control of heart rate do not occur in conscious humans with changes in central venous pressure within a physiological range. These results are consistent with studies in humans and monkeys which showed no tachycardia during immersion (1,16) or rapid intravenous infusion (8,30) despite substantial increases in central venous pressure. The difference between the results in dogs and the primate may reflect species difference in the role of cardiopulmonary baroreceptors in control of circulation.

In summary, our study suggests that variations of central venous pressure and cardiopulmonary receptor activity within a physiological range do not modulate sinus node responses to arterial hypertension in man. We have not excluded the possibility that cardiopulmonary receptor activity modifies sinus node responses to arterial hypotension, or that there might be an

interaction between cardiopulmonary baroreceptor activity and arterial baroreflex control of heart rate when central venous pressure is elevated above the levels in our study.

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LEGEND

Figure 1: Effect of propranolol on the slope of baroreflex control of R-R interval. The ** indicates $p < 0.01$.

Sinus node responses to baroreflex stimulation were greater after propranolol than before, but the slopes after propranolol were comparable during lower body negative pressure, the control state, and elevation of the trunk and legs.

Figure 2: Prolongation of the R-R interval by 30 mmHg neck suction during the control state; the early dynamic phase; steady-state; and offset of changes of central venous pressure produced 20 mmHg lower body negative pressure and elevation of the trunk and legs. Sinus node responses to neck suction were comparable during all interventions.

FOURTH EDITION

Hemodynamic Findings and Arterial Baroreflex

Subject	Control Before LBNP					LBNP at 20 mmHg				
	HR (beats/ min)	SAP (mmHg)	CVP (mmHg)	FoBF (ml/min /100gm)	Baroreflex Slope (msec/mmHg)	HR (beats/ min)	SAP (mmHg)	CVP (mmHg)	FoBF (ml/min /100gm)	Bar S (msec/mmHg)
CY	70	108	2.2	4.8	16.7	72	108	-2.5	2.4	
DW	42	125	6.5	3.4	23.7	43	130	1.5	2.7	
AT	79	140	5.5	5.6	6.8	83	133	1.5	3.8	
BM	58	110	5.0	5.3	14.4	57	123	0	3.1	
DB	72	132	6.5	5.1	8.7	63	135	1.0	2.8	
MM	72	103	6.0	4.6	14.9	72	105	0	3.3	
MR	58	116	10.0	3.2	11.6	60	123	6.0	2.4	
mean	64	119	6.0	4.6	13.8	64	122	1.1*	2.9*	
SE	5	5	0.9	0.3	2.1	5	5	1.0	0.2	

LBNP: lower body negative pressure

HR: heart rate

SAP: systolic arterial pressure

CVP: central venous pressure

FoBF: forearm blood flow

*: p < 0.01 LBNP vs Control Before LBNP

†: p < 0.01 Elevation of Legs and Trunk vs Control Before Legs and Trunk Elevation

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Table 1

Reflex Responses During Changes in Central Venous Pressure

Baroreflex Slope (msec/mmHg)	Control Before Leg and Trunk Elevation					Elevation of Legs and Trunk				
	HR (beats/ min)	SAP (mmHg)	CVP (mmHg)	FOBF (ml/min /100gm)	Baroreflex Slope (msec/mmHg)	HR (beats/ min)	SAP (mmHg)	CVP (mmHg)	FOBF (ml/min /100gm)	Baroreflex Slope (msec/mmHg)
15.7	68	111	2.0	4.0	16.7	66	113	5.0	4.0	18.2
25.2	41	135	5.0	3.4	21.7	42	140	9.0	3.4	24.9
7.3	84	138	5.0	5.1	8.4	76	142	7.5	5.0	6.9
14.4	52	127	5.0	4.1	15.4	53	128	7.5	4.5	14.8
10.4	61	132	6.5	5.4	9.3	63	135	10.0	6.7	11.0
15.9	--	---	---	---	---	68	115	9.0	5.3	15.4
10.9	--	---	---	---	---	60	128	15.0	5.0	6.0
14.3	61	129	4.7	4.4	14.3	61	129	9.0†	4.8	13.9
2.2	7	4	0.7	0.4	2.5	4	4	1.2	0.4	2.5

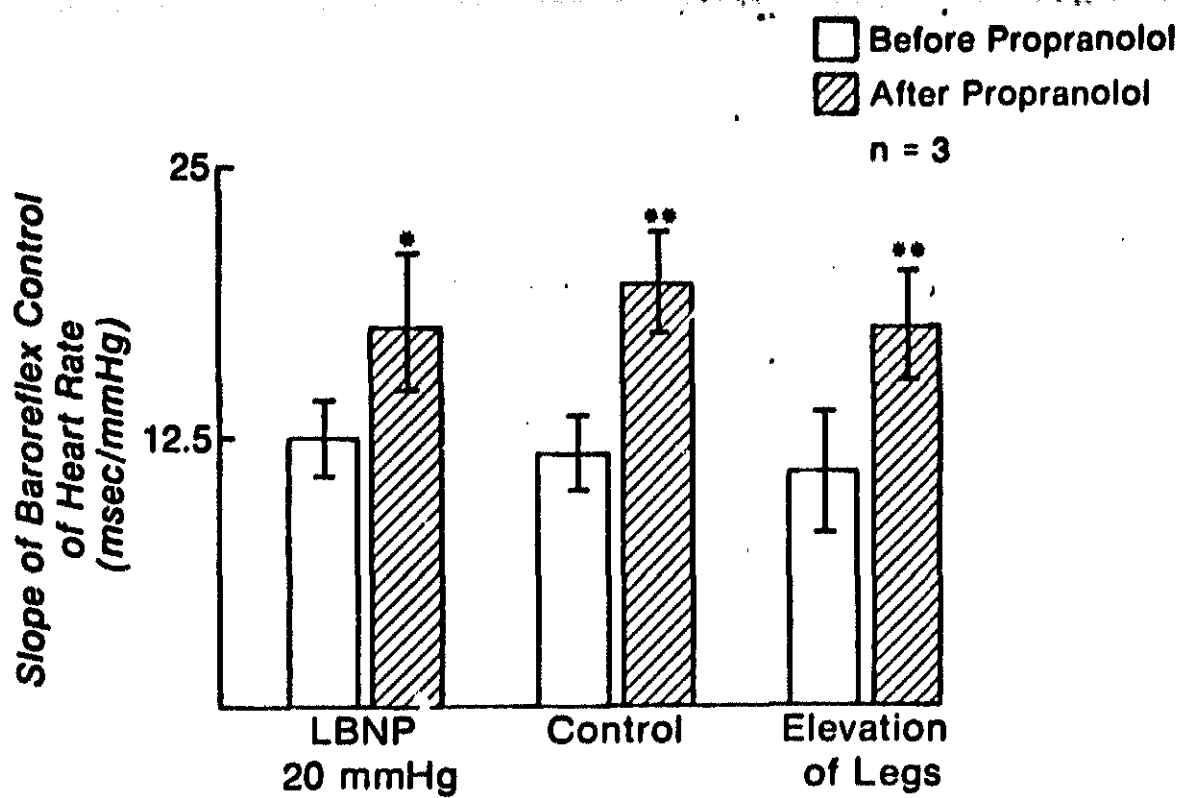


Fig. 1

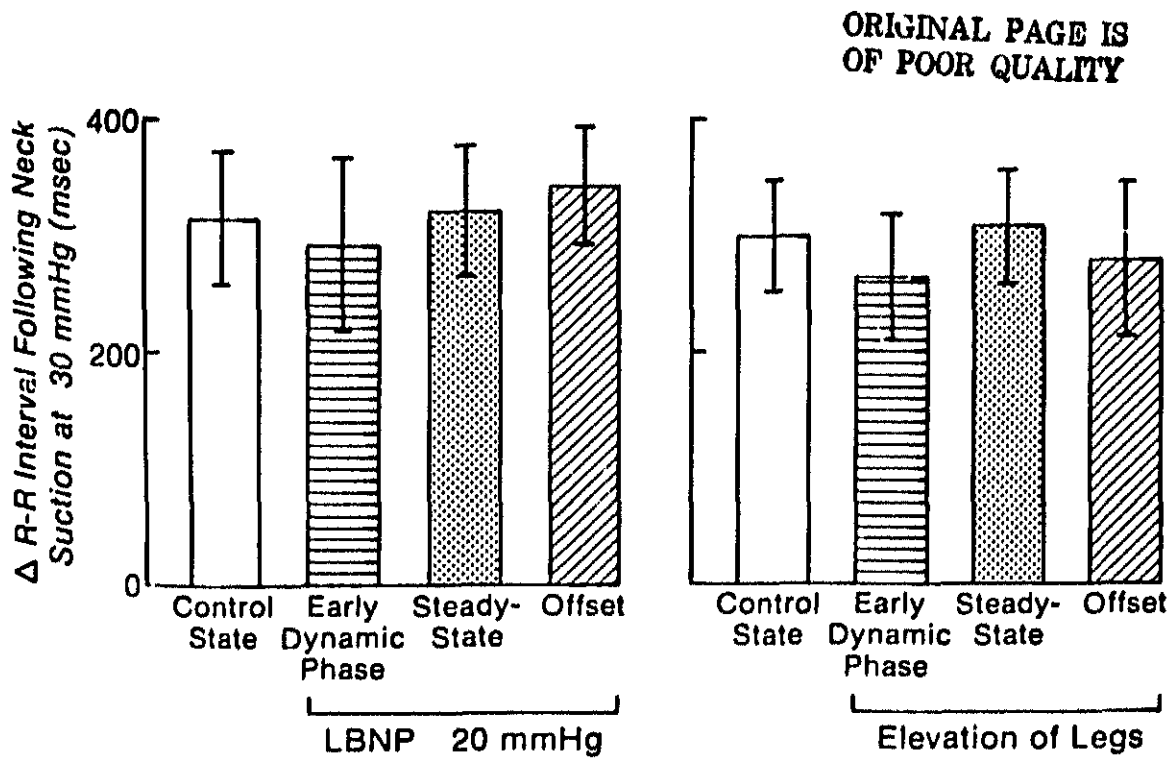


Fig. 2